Amendment to the Specification

In the specification, on page 4, the paragraph in lines 16-22 shall be deleted, and replaced by the following replacement paragraph.

It is a further object of the present invention to provide methods for treating an individual with a composition for depleting B cells, including to a subpopulation of B cells which are activated by shed antigen released from CNS tissue damaged by an inflammatory process, which B cells may also be found circulating in body fluids selected from the group consisting of peripheral blood, cerebrospinal fluid, and a combination thereof (as disclosed in ~~co-pending U.S. Serial No. 60/150,256~~ U.S. Patent No. 6,762,032), wherein the depletion of B cells reduces inflammation which causes clinical manifestations associated with progressive MS.

In the specification, the paragraph beginning at line 7 on page 13 and ending at page 14, line 7 shall be deleted, and replaced by the following replacement paragraph.

The term "pro-MS immune response" is used herein, for purposes of the specification and claims, to mean a humoral immune response induced against an epitope comprising a terminal alpha 2,6 linked sialic acid (e.g., comprising sialyl Tn or sTn which comprises a terminal sialic acid alpha 2,6 linked to Ga1NAc; or alternatively, to Ga1) of a shed antigen (glycomolecule), resulting in production of IgG antibody against the epitope ("anti-α (2,6) NeuAc Ab"), and complexes comprised of the shed antigen comprising the epitope complexed to anti-α (2,6) NeuAc Ab; wherein the shed antigen is released or produced particularly in relation to CNS tissue damage characteristic of MS during the a chronic inflammatory disease process characteristic of inflammatory forms of MS (e.g., secondary progressive MS). In a preferred embodiment, the resultant complexes bind to and induce Fc receptor-expressing cells (e.g., one or more cell types selected from the group consisting of granulocytes, macrophages, microglia, activated mast cells, astrocytes, oligodendrocytes) which results in the release of inflammatory mediators (e.g., cytokines and/or tissue degradative enzymes) which may exacerbate

the existing inflammatory process and thereby promote (contribute to) CNS tissue damage characteristic of MS (e.g., demyelination and plaques characteristic of MS). A similar immune response, a pro-tumor immune response, and it's ability to promote inflammation and tissue degradation has been described in ~~co-pending U.S. application Ser. No. 09/435,289~~ U.S. Patent No. 6,251,616 (the disclosure of which is herein incorporated by reference). In a preferred embodiment, the anti-α (2,6) NeuAc Ab is induced by a shed antigen comprising glycolipid; and in a more preferred embodiment, glycolipid selected from one or more of the alpha series of gangliosides (e.g., GD1α, GT1aα, GQ1bα, derivatives thereof which contain one or more additional terminal sialic acids alpha 2,6 linked to Ga1NAc, and a combination thereof). Serological markers for a pro-MS immune response have been described in detail in ~~co-pending U.S. applications~~